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"INTRACEREBRAL HEMORRHAGE IN THE YOUNG."[1]

BY B. SACHS, M.D.

Within the last few years, the pathology of the infant's brain has attracted unusual attention. As a result of closer study, we have learned to differentiate between a number of forms of paralysis which were at one time included under the comprehensive term of 'infantile paralysis.' In this connection, I need merely remind you of our increased knowledge on polioencephalitis to be ascribed mainly to the careful researches of Kundrat, Henoch, Strümpell, Bernhardt, and in no mean degree to an excellent paper by Dr. McNutt. With polioencephalitis I am in no wise concerned in this paper. Leaving aside paralyses to be ascribed to this condition, I am safe in saying that suddenly developed paralyses, and hemiplegiæ in particular, have been too frequently attributed to meningeal hemorrhage. No doubt that in most, and in traumatic cases particularly, an effusion of blood into the meninges or over the cortex is the cause of the paralysis. It is my own conviction, however, that intracerebral hemorrhage is more frequent in the young than it is generally supposed to be. If so, it will be important to be able to make a differential diagnosis between these two forms of intracranial hemorrhage at a very early stage of the trouble, or rather between hemiplegia due to meningeal, and hemiplegia due to intracerebral, hemorrhage. If for no other reason, it will be important in order to give a correct prognosis.

I have said that I believed intracerebral hemorrhage to be more frequent in the young than it was generally considered to be. Withal, the condition is rare enough to merit full discussion, and I have therefore no hesitation in calling your attention to a few cases of this type.

I wish to anticipate criticism of my remarks by confessing that I have no autopsy to prove my diagnosis, and that I may be guilty of what Dr. Seguin once styled 'speculative pathology.' But

[1] This paper, although read a year ago at the meeting of the Am. Neurol. Association, has not hitherto been published, as the author wished to confirm his views by autopsies. In view of the appearance of Dr. Knapp's paper, the author has determined to publish these "clinical cases." Case I. has since died of malignant diphtheria, but unfortunately no post-mortem examination was granted.

there are conditions which rarely lead to the post-mortem table and yet are of commanding interest. Moreover, in the present state of cerebral pathology we are, I claim, fully able to infer pathologicial appearances from clinical data. The clinical data, and the inferences drawn therefrom, must be beyond dispute. You will find a close resemblance between the histories here presented to you and that of an ordinary adult apoplexy. This striking analogy may be taken as one proof of the identity of the pathological processes. The details of my first case are as follows:

CASE I.—One of three children; good family history; no syphilis.

The patient, M. M., is a healthy-looking boy two and a half years old. Up to the time of present illness in October, 1885, boy had had no other diseases but chicken-pox, eczema, and one and a half years previously, inflammation of the lungs. No history of fall. October 24th I was called to see the child and was told that it had been restless the night before, that it had vomited once, and seemed to have some difficulty in swallowing its food. Inspection showed that the tonsils were considerably swollen, the tongue slightly coated, but there was nothing of a diphtheritic character visible on or about the tonsils. The rectal temperature was 101.4.° During the next few days the tonsillar symptoms abated under the ordinary iron treatment. The temperature never exceeded 102°, and the pulse varied between 90 and 110. I saw the child on the morning of the 27th, found all its symptoms in abeyance, the child moved about freely, was feeling well and answered questions in its childish way, played with its toys, and did not seem in need of further medical attendance.

Early the following morning, the father called upon me in great distress, saying that his child could not speak and that it was not able to move its right side. My first fear was that the throat trouble had perhaps been of a more serious character, and that the child was now afflicted with a form of diphtheritic paralysis; but on seeing the patient I rid myself very quickly of this notion, for to my knowledge diphtheritic paralysis is never of the hemiplegic order.

At the patient's house I learned the following: On the day previous, soon after I had left the child, the latter seemed to grow very quiet; the nurse was struck by the fact that the child had given up calling her name dozens of times in succession as had been its habit; she did not remember having heard a single word pass the child's lips since that morning. No further attention was paid to the matter, as the mother and nurse thought the child sullen and nothing more. The nurse claims that the child used its arms perfectly well during the day, that it climbed of its own accord from one bed into another about 8 o'clock in the evening. At about 10 o'clock the mother noticed that the child moved its left arm and not its right, and the nurse noticed during the night that the child embraced her with its left hand only, that it lay on

its right side and did not seem able to change its position; the nurse turned the child several times during the night. Loss of consciousness was not observed at any time, although it is very evident that some paralysis had set in before the child fell asleep. The child was watched continuously day and night, and no convulsive movements of any sort, universal or partial, were observed.

On examining the child, I was greatly surprised to find typical right hemiplegia with aphasia—if you will allow the bull—adult hemiplegia in a child two and a half years old. I found that while the child seemed to understand all that was said to it, it could not be made to utter a single word. It moved its lips as though it wished to speak but could not. I thought, as I was about to leave the room, that it said good-by; but in this I may have been deceived, for the other persons in the room did not hear it, and certain it is that during the remainder of the day it did not utter a single word.

The right arm and right leg were markedly paretic, not absolutely paralyzed. It could move its hand a little, both spontaneously and when asked to do so; when lifted, the arm did not fall entirely "dead" upon the bed. The child was placed upon its legs for an instant, when it was evident that the right foot was dragged and that it made no independent movements. When the child was put to bed, I found that the right leg was slowly withdrawn after tickling the sole, but not after pinching the leg. There was slight and facial paresis. The knee-jerks could be elicited on both sides. Sensation normal in every respect, ocular movements were perfect; pupils equal and contracted promptly; water and fæces were passed voluntarily. The symptoms remained unchanged during the day, except that in the evening the child seemed less bright than in the morning. It did everything, however, which it was asked to do, showed its tongue, took its food, and so on.

Evening temperature in rectum 100°, pulse 102°. Heart sounds normal, optic discs ditto, urine free from albumin and sugar. No vomiting at any time.

During the next four days the condition remained practically unchanged. The child did not utter a word, but could be made to laugh when his little brother spoke to him. On the fourth day, there was a slight improvement in the movement of the leg, but none in those of the arm. The treatment consisted at first in small doses of sodium iodide, and ergot. This was soon abandoned for inunctions of the oleate of mercury. Absolute rest, ice to head and neck were the only other remedial measures employed.

On the fifth day the improvement was more decided; the child said "papa" when its father came near the bed, and spoke the end words of some little rhymes that it was accustomed to repeat when well. The right foot could be drawn up some distance, under the influence of a stronger incentive (holding its toe back);

the arm has not perceptibly improved; child laughs heartily, and when doing so the right facial paresis is more evident than when the face is at rest; it does not attempt to blow at watch; plantar reflexes equal; knee joints ditto. Sensation continued normal.

The further history of the case need not be given in full. The child recovered as adult hemiplegics do; first the use of the leg and then of the arm. On the thirteenth day after initial symptoms was able to stand on its legs unsupported. From the eighteenth day on, it began using its hand, scribbling all day long. From the thirteenth day on, began to speak in sentences. After three weeks all symptoms had disappeared, and during the past eight months the child has exhibited no other cerebral symptoms; but it has at present the whooping cough, the very trouble I feared most. Thus far it has done well and has escaped all further cerebral trouble.

On the third day, I made the diagnosis of intracerebral hemorrhage, probably hemorrhage into lenticular nucleus; prognosis was favorable from the outset.

The earlier history of the case I gave in great detail, in order to put the diagnosis upon a firm basis. The resemblance to ordinary adult apoplexy is so great that we might argue about the symptoms by analogy only; but I prefer to examine them in unbiased fashion. Two facts stand out most prominently; these are, no loss of consciousness and entire absence of convulsions. How do these symptoms bear upon the differential diagnosis; first, as regards the immediate cause of the apoplexy (using that word in its broadest sense), and secondly, as regards the differential diagnosis between meningeal and cerebral hemorrhages? Was the hemiplegia due to thrombosis, embolism, or hemorrhage? There are no facts which argue in favor of thrombosis. As for embolism, there is little in support of that possibility. There is no cardiac mischief, no history of rheumatism, and the mode of onset was slow, not sudden. If the history be correct, as given above, the child felt ill at ease during an entire day before the attack developed. The aphasia was developed first, the paralysis of arm and leg several hours later. There is but one statement, made by no less an authority than Nothnagel, which might lead us to give a different interpretation to these facts. In the article on cerebral hemorrhage in Ziemssen's large work, Nothnagel, discussing the differential diagnosis between embolism and hemorrhage, declared that if hemiplegia be recovered from within a few days this would argue rather against hemorrhage and in favor of embolism. This statement is misleading, for we must not forget that there are direct and indirect lesion symptoms—a fact which Nothnagel in later years helped to prove. Hemorrhage into the lenticular nucleus is a very different occurrence from hemorrhage into the internal capsule: in the one case the hemorrhage is adjacent to, in the other it immediately involves the greater part of the motor tract; in the one case it

might suspend function temporarily, in the other it would interfere with motor functions for a long period of time, according to the amount of damage originally done, the power of recovery of the parts involved, or the power of other parts to assume the function of those destroyed. It is well known, also, that embolism is far more frequently accompanied by loss of consciousness than hemorrhage. The plugging of even a very small cerebral artery is almost invariably followed by loss of consciousness. In this case, not the amount of injury done, but the suddenness with which it is inflicted seems to be the more important factor. While there is little or no reason to suppose that the hemiplegia is due to embolism, there is everything in favor of hemorrhage except the age of the patient.

The mode of onset was such as we should expect from slow and small hemorrhage. There was no loss of consciousness, and there was prompt recovery in the course of a few weeks. What the conditions are causing such hemorrhage we shall discuss shortly.

But might this not after all have been a case of meningeal hemorrhage; can we decide the question definitely? I repeat the close clinical analogy between this and adult cases would justify us in supposing the pathological processes to be similar, and in adults we know this form of apoplexy to be due almost invariably to intracerebral hemorrhage. Does the preservation of consciousness during the onset of the attack or the absence of convulsions help in making a differential diagnosis between meningeal and intracerebral hemorrhage? Loss of consciousness is an extremely variable symptom; it seems to depend rather upon the quantity of blood effused than upon the area involved. Not so with convulsions. A convulsion, if it is anything, is a cortical affair, the result of cortical irritation; small hemorrhage over or near the cortex will be apt to bring on convulsions, and this may be confined strictly to the motor portions of one-half of the cortex. Convulsions are the invariable accompaniment of meningeal hemorrhage. An examination of a large number of cases of meningeal hemorrhage in dispensary practice brings out this fact very distinctly.

Very large intracerebral hemorrhage might possibly produce convulsions in children, but the absence of such convulsions argues, to my mind, strongly in favor of intracerebral hemorrhage and against meningeal hemorrhage. The symptoms of both may resemble each other in every other respect; hence the importance of paying special attention to this point. The prognosis in intracerebral hemorrhage is certainly far more favorable, and in my own case the absence of convulsions enabled me on the third day after the attack to prognosticate complete recovery.

In the early stages of acute polioencephalitis the symptom may in some respects resemble those discussed here. That is, the polioencephalitis may give rise to hemiplegic symptoms, but in my

case the preservation of consciousness, the lack of convulsions and the prompt recovery above all things, argue against the presence of polioencephalitis.

The rarity of intracerebral hemorrhage in the young, in fact, in persons under forty, is acknowledged by all, and in the case which I wish to refer to now the diagnosis was considered uncertain by several neurologists, and yet this patient was nearly seventeen years older than my first case.

CASE II. I had the privilege of seeing in private practice only a short time ago. The patient is a young man about nineteen years of age, with good family history, of excellent habits, and without hereditary or acquired syphilis. He is by no means frail in appearance, though rather undersized, and his father states that the boy stopped growing suddenly. About a year and one-half ago he was preparing very earnestly for his college examinations, when he was seized with a slight left hemiplegia. The onset was gradual, there were neither coma nor convulsions; and after three weeks, during which the symptoms had receded typically, he had completely recovered. He went along very well for exactly one year when one day, as he was paying a visit, he felt numbness coming on as it did in the previous attack. He had courage enough to start for home; when he arrived there he began to vomit, became comatose (one and one-half hours after initial numbness) and remained in this state of coma for eighteen hours; no convulsions. He had complete left hemiplegia, but no aphasia although he was congenitally left-handed; right-handed by education.

The paralysis remained unchanged for a fortnight. Recovery began with leg; now he has full use of his lower extremities, though he drags his left foot in characteristic fashion. He has exaggerated knee-jerks on both sides, and very marked double ankle clonus; his left hand is still paretic and contractured, no cardiac or renal trouble. The diagnosis reads intracerebral hemorrhage, involving destruction of a large part of the internal capsule. In this case, mental overwork is considered the direct exciting cause; in the first case, no direct cause could be made out. The child had no acute infectious or exhausting disease, and had not at that time the whooping cough—causes to which intracerebral hemorrhage in the young has been ascribed by writers on the subject. I cite the second case to show the analogous symptoms in the two. In both hemorrhage occurred in persons far below the usual age limit; in both syphilitic changes can be safely excluded. Convulsions were absent in both, while in the one case coma supervened during one of the two attacks.

The second case proves the danger of this tendency to hemorrhage when exhibited in very young persons—the danger of repeated attacks. In this regard the prognosis must be held to be unfavorable.

And now as to the pathology of the two cases. This is at once

the most interesting and the least satisfactory aspect of the entire subject. The analogy with adult cases would lead us to suppose that hemorrhages in these cases were due to the rupture of miliary aneurisms of the middle cerebral artery. The probability is very strong, but the reports of post-mortem examinations which showed intracranial (including meningeal) hemorrhage in young persons to be due to the rupture of small aneurisms are exceedingly few. In his classical work on the "Pathology of the Circulation and Nutrition," Recklinghausen states (p. 84) that he has seen but a single fatal case of intracerebral hemorrhage in the young, and this was a case of chronic renal disease with hypertrophy of the heart. In this connection I may refer to a publication of Dr. Osler in the Canada *Medical and Surgical Journal* (1886), in which he reports the finding of an aneurism of a branch of the anterior cerebral in a boy six years of age; as Dr. Osler adds, to his knowledge the youngest case on record.[1] In view of this paucity of knowledge on this subject, we must stop to inquire whether hemorrhage may not be due to other conditions than those of aneurism? There is but one change in the blood-vessels of the young which is familiar to many pathologists, and that is a fatty degeneration of the walls of smaller blood-vessels, and again Recklinghausen suggests that there may be a simple transudation of blood through these pathologically altered blood-vessels. The pathology of the vascular system of the young is a subject of surpassing interest. It is almost virgin soil on which some of us should plough successfully.

[1] Boy, aged 6, brought to hospital unconscious with feeble pulse, pale face, eye's and head turned to right, and left hemiplegia. Death in six hours. He had fallen from a hay loft three weeks before, but he recovered rapidly from the effects. There was meningeal hemorrhage at base and in longitudual fissure. An aneurismal sac was found imbedded in the calloso-marginal fissure just where it turns vertically upwards. The rupture was on the meningeal surface, but hemorrhage had extended into contiguous portion of brain. The arteries were not atheromatous, presumably altogether normal, and the heart was healthy.